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(VASCULAR MEDICINE (NON-CORONARY): CLINICAL SCIENCE)

41. The myocardial protective effect of dexmedetomidine in high risk patients undergoing aortic vascular surgery

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Dexmedetomidine provides perioperative cardiac protection in high risk patients assessment the effect of dexmedetomidine in high risk patients undergoing aortic vascular surgery. Arandomized study included 150 patients classified into two groups($n = 75$). Group D: The patients received a loading dose of 1 μg/kg dexmedetomidine over 15 min before induction and maintained as an infusion of 0.3 μg/kg/hr to the end of the procedure. Group C: The patients received an equal volume of normalsaline. The dexmedetomidine decreased heart rate and minimized the changes in blood pressure compared to control group ($p < 0.05$). Also, it decreased the incidence of myocardial is chemia reflected by troponin I level and ECG changes($p < 0.05$). Dexmedetomidine decreased the requirement for nitroglycerine and norepinephrine compared to control group ($p < 0.05$). The incidence of hypotension and bradycardia were significantly higher with dexmedetomidine ($p < 0.05$). The dexmedetomidine is safe and effective in patients undergoing aortic vascular surgery. It decreases the changes in heart rate and blood pressure during the procedures. It provide scardiac protection in high risk patients reflected by decreasing the incidence of myocardial ischemia and serum level of troponin. The main side effects of dexmedetomidine were hypotension and bradycardia.

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Microcirculation and Cerebral/Coronary/peripheral circulation

42. The effect of ticagrelor on coronary blood after primary PCI when compared with clopidogril

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Primary PCI (PPCI) has been established as the best treatment for acute MI when it is used appropriately. It is known to give better TIMI III flow and better frame count when compared with thrombolytics. Loading with P2Y12 inhibitors in the ER prior to primary PCI is an important step in antiplatelet therapy for acute myocardial infarction. In this study We report the effect of loading with two different P2Y12 inhibitors (ticagrelor and clopidogril) on the TIMI frame count in the culprit artery after successful PPCI. Ticagrelor may affect coronary microcirculation and coronary blood flow through faster and stronger platelet inhibition. We randomized 44 patient who presented to our center with acute MI into two groups. The first group received a loading with 180mg of ticagrelor and the second group received a loading with 600 mg of clopidogril. The mean door to balloon time was 98 ± 12 min. All patients in both groups received a loading with 162 mg of aspirin. GP IIb/IIIa inhibitors were used in all cases together with adjusted dose heparin. Stent usage was 100%. No thrombectomy or thrombus aspiration device was used in any of these cases. TIMI III flow after stenting was achieved in all culprit arteries. Then we calculated the TIMI frame count in the culprit artery after successful primary PCI. The mean corrected TIMI frame count in the culprit artery post PCI was 18.34 ± 3.16 frames in group 1 (Ticagrelor group) and 28.73 ± 3.92 in group 2 (clopidogril group) ($p = 0.02$). Loading with ticagrelor gives faster flow after successful primary PCI in the culprit artery of acute MI when compared with clopidogril. This can be explained by the fact that ticagrelor therapy give faster P2Y12 inhibition thus faster antiplatelet therapy causing less platelet aggregation resulting in less distal embolization and reduced production of inflammatory mediators and adhesion molecules which may result in faster restoration of normal endothelial function. This finding may partially explain the mortality benefit of ticagrelor in a previous ACS study. A larger prospective randomized study is needed to confirm this finding.

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Cellular biology function

LIGAND-MEDIATED SIGNALING AND RECEPTOR PHARMACOLOGY

43. Calmodulin regulating calcium sensitivity of Na channels

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By extrapolating information from existing research and observing previous assumptions regarding the structure of the Na Channel, this experiment was conducted under the hypothesis that the Na Channel is in part regulated by the calmodulin protein, as a result proving calcium sensitivity of the Na Channel. Furthermore, we assume that there is a one to one stoichiometry between the Na Channel and the Calmodulin. There has been extensive research into the functionality and structure of sodium ion channels (Na channels), as several diseases are associated with the lack of regulation of sodium ions, that is caused by the disfunction of these Na channels. However, one highly controversial matter in the field is the importance of the protein calmodulin (CaM) and calcium in Na channel function. Calmodulin is a protein that is well known for its role as a calcium binding messenger protein, and that association is believed to play an indirect role in regulating the Na chan-